THE HEALTH-SUPPORTING BENEFITS OF COCONUTS

Coconuts and coconut oil contain health-promoting saturated fatty acids and derivative compounds which have powerful antimicrobial properties.

Part 1 of 2

by Mary G. Enig, PhD, FACN

Director
Nutritional Sciences Division
Enig Associates, Inc.
12501 Prosperity Drive, Suite 340
Silver Spring, MD 20904-1689, USA
Telephone: +1 (301) 680 8600
Fax: +1 (301) 680 8100
Email: marye@enig.com

The following is the text of a talk and paper, "Coconuts: In Support of Good Health in the 21st Century", presented by Dr Mary Enig at the Asian Pacific Coconut Community (APCC) meeting held in Pohnpei in the Federated States of Micronesia in 1999. Note that it does make several references to animal experiments, and that* NEXUS *does not condone animal experimentation. Editor.

ABSTRACT

Coconuts play a unique role in the diets of mankind because they are the source of important physiologically functional components. These physiologically functional components are found in the fat part of whole coconut, in the fat part of desiccated coconut and in the extracted coconut oil.

Lauric acid, the major fatty acid from the fat of the coconut, has long been recognised for the unique properties that it lends to nonfood uses in the soaps and cosmetics industry. More recently, lauric acid has been recognised for its unique properties in food use, which are related to its antiviral, antibacterial and antiprotozoal functions. Now, capric acid, another of coconut's fatty acids, has been added to the list of coconut's antimicrobial components. These fatty acids are found in the largest amounts only in traditional lauric fats, especially from coconut. Also, recently published research has shown that natural coconut fat in the diet leads to a normalisation of body lipids, protects against alcohol damage to the liver and improves the immune system's anti-inflammatory response.

Clearly, there has been increasing recognition of the health-supporting functions of the fatty acids found in coconut. Recent reports from the US Food and Drug Administration about required labelling of the *trans* fatty acids will put coconut oil in a more competitive position and may help its return to use by the baking and snack-food industry, where it has continued to be recognised for its functionality. Now it can be recognised for another kind of functionality: the improvement of the health of mankind.

I. INTRODUCTION: BENEFITS OF COCONUT OIL SATURATES

Mr Chairman and members of the Asian Pacific Coconut Community: I would like to thank you for inviting me once again to speak to this gathering of delegates on the occasion of your 36th session as you celebrate the 30th anniversary of APCC.

When I addressed the 32nd Cocotech meeting in Cochin, India, I covered two areas of interest to the coconut community. In the first part, I reviewed the major health challenge facing coconut oil at that time, which was based on a supposed negative role played by saturated fat in heart disease. I hope that my talk was able to dispel any acceptance of that notion. In the second part of my talk, I suggested that there were some new, positive health benefits from coconut which should be recognised. These benefits stemmed from coconut's use as a food with major functional properties for antimicrobial and anti-cancer effects.

In my presentation today, I will bring you up to date about the new recognition of "functional foods" as important components in the diet. Additionally, I would like to review briefly the state of the anti–saturated fat situation and bring you up to date on some of the research that compares the beneficial effects of saturated fats with those of omega-6 polyunsaturates, as well as the beneficial effects of the saturated fats relative to the detrimental effects of the partially hydrogenated fats and the *trans* fatty acids. In particular, I will review some of the surprising beneficial effects of the special saturates found in coconut oil as they compare with those of the unsaturates found in some of the other food

oils. Components of coconut oil are increasingly being shown to be beneficial. Increasingly, lauric acid and even capric acid have been the subject of favourable scientific reports on health parameters.

II. FUNCTIONAL PROPERTIES OF LAURIC FATS AS ANTIMICROBIALS

Earlier this year, at a special conference entitled "Functional Foods For Health Promotion: Physiologic Considerations" (Experimental Biology '99, Renaissance Washington Hotel, Washington, DC, April 17, 1999), which was sponsored by the International Life Sciences Institute (ILSI) North America, Technical Committee on Food Components for Health Promotion, it was defined that "a functional food provides a health benefit over and beyond the basic nutrients".

This is exactly what coconut and its edible products such as desiccated coconut and coconut oil do. As a functional food, coconut has fatty acids that provide both energy (nutrients) and raw material for antimicrobial fatty acids and monoglycerides (functional components) when it is eaten. Desiccated coconut is about 69% coconut fat, as is creamed coconut. Full coconut milk is approximately 24% fat.

Approximately 50% of the fatty acids in coconut fat are lauric acid. Lauric acid is a medium-chain fatty acid which has the additional beneficial function of being formed into monolaurin in the human or animal body. Monolaurin is the antiviral, antibacterial and antiprotozoal monoglyceride used by the human (and animal) to destroy lipid-coated viruses such as HIV, herpes, cytomegalovirus, influenza, various pathogenic bacteria including *Listeria monocytogenes* and *Helicobacter pylori*, and protozoa such as *Giardia lamblia*. Some studies have also shown some antimicrobial effects of the free lauric acid.

Also, approximately 6–7% of the fatty acids in coconut fat are capric acid. Capric acid is another medium-chain fatty acid which has a similar beneficial function when it is formed into monocaprin in the human or animal body. Monocaprin has also been shown to have antiviral effects against HIV and is being tested for antiviral effects against herpes simplex and for antibacterial effects against *Chlamydia* and other sexually transmitted bacteria (Reuters, London, June 29, 1999).

The food industry has, of course, long been aware that the functional properties of the lauric oils, and especially coconut oil, are unsurpassed by other available commercial oils. Unfortunately in the United States, during the late 1930s and again during the 1980s and 1990s, the commercial interests of the domestic fats and oils industry were successful in driving down usage of coconut oil. As a result, in the US and in other countries where the influence from the US is strong, the manufacturer has lost the benefit of the lauric oils in its food products.

As we will see from the data I will present in this talk, it is the consumer who has lost the many health benefits that can result from regular consumption of coconut products.

Unfortunately in the United States, during the late 1930s and again during the 1980s and 1990s, the commercial interests of the domestic fats and oils industry were successful in driving down usage of coconut oil.

The antiviral, antibacterial and antiprotozoal properties of lauric acid and monolaurin have been recognised by a small number of researchers for nearly four decades. This knowledge has resulted in more than 20 research papers and several US patents, and last year it resulted in a comprehensive book chapter which reviewed the important aspects of lauric oils as antimicrobial agents (Enig, 1998). In the past, the larger group of clinicians and food and nutrition scientists has been unaware of the potential benefits of consuming foods containing coconut and coconut oil, but this is now starting to change.

Kabara (1978) and others have reported that certain fatty acids (FAs) (e.g., medium-chain saturates) and their derivatives (e.g., monoglycerides, MGs) can have adverse effects on various micro-organisms. Those micro-organisms that are inactivated include bacteria, yeast, fungi and enveloped viruses. Additionally, it is reported that the antimicrobial effects of the FAs and MGs are additive, and total concentration is critical for inactivating viruses (Isaacs and Thormar, 1990).

The properties that determine the anti-infective action of lipids are related to their structure, e.g., monoglycerides, free fatty acids. The monoglycerides are active; diglycerides and triglycerides are inactive. Of the saturated fatty acids, lauric acid has greater antiviral activity than caprylic acid (C-8), capric acid (C-10) or myristic acid (C-14).

In general, it is reported that the fatty acids and monoglycerides produce their killing/inactivating effect by lysing the plasma membrane lipid bilayer. The antiviral action attributed to monolaurin is that of solubilising the lipids and phospholipids in the envelope of the virus, causing the disintegration of the virus envelope. However, there is evidence from recent studies that one antimicrobial effect in bacteria is related to monolaurin's interference with signal transduction (Projan et al., 1994), and another antimicrobial effect in viruses is due to lauric acid's interference with virus assembly and viral maturation (Hornung et al., 1994).

Recognition of the antiviral aspects of the antimicrobial activity of the monoglyceride of lauric acid (monolaurin) has been reported since 1966. Some of the early work by Hierholzer and Kabara (1982), which showed virucidal effects of monolaurin on enveloped RNA and DNA viruses, was done in conjunction with the Centers for Disease Control of the US Public Health Service. These studies were done with selected virus prototypes or recognised representative strains of enveloped human viruses. The envelope of these viruses is a lipid membrane, and the presence of a lipid membrane on viruses makes them especially vulnerable to lauric acid and its derivative, monolaurin.

The medium-chain saturated fatty acids and their derivatives act by disrupting the lipid membranes of the viruses (Isaacs and Thormar, 1991; Isaacs et al., 1992). Research has shown that enveloped viruses are inactivated in both human and bovine milk by added fatty acids and monoglycerides (Isaacs et al., 1991) and also by endogenous fatty acids and monoglycerides of the

appropriate length (Isaacs et al., 1986, 1990, 1991, 1992; Thormar et al., 1987).

Some of the viruses inactivated by these lipids, in addition to HIV, are the measles virus, herpes simplex virus-1 (HSV-1), vesicular stomatitis virus (VSV), visna virus and cytomegalovirus (CMV). Many of the pathogenic organisms reported to be inactivated by these antimicrobial lipids are those known to be responsible for opportunistic infections in HIV-positive individuals. For example, concurrent infection with cytomegalovirus is recognised as a serious complication for HIV-positive individuals (Macallan et al., 1993).

Thus, it would appear to be important to investigate the practical aspects and the potential benefits of an adjunct nutritional support regimen for HIV-infected individuals, which will utilise those dietary fats that are sources of known antiviral, antimicrobial and antiprotozoal monoglycerides and fatty acids such as monolaurin and its precursor, lauric acid.

Components of coconut oil, such as lauric acid, are increasingly being shown to be beneficial.

Until now, no one in the mainstream nutrition community seems to have recognised the added potential of antimicrobial lipids in the treatment of HIV-infected or AIDS patients. These antimicrobial fatty acids and their derivatives are essentially nontoxic to man; they are produced *in vivo* by humans when they ingest those commonly available foods that contain adequate levels of medium-chain fatty acids such as lauric acid. According to the published research, lauric acid is one of the best "inactivating" fatty acids, and its monoglyceride is even more effective than the fatty acid alone (Kabara, 1978; Sands et al., 1978; Fletcher et al., 1985; Kabara, 1985).

The lipid-coated (enveloped) viruses are dependent on host lipids for their lipid constituents. The variability of fatty acids in the foods of individuals, as well as the variability from *de novo* synthesis, accounts for the variability of fatty acids in the virus envelope and also explains the variability of glycoprotein expression—a variability that makes vaccine development more difficult.

Monolaurin does not appear to have an adverse effect on desirable gut bacteria but, rather, only on potentially pathogenic micro-organisms. For example, Isaacs et al. (1991) reported no inactivation of the common *Escherichia coli* or *Salmonella enteritidis* by monolaurin, but major inactivation of *Hemophilus influenzae*, *Staphylococcus epidermidis* and group B gram-positive *Streptococcus*.

The potentially pathogenic bacteria inactivated by monolaurin include *Listeria monocytogenes*, *Staphylococcus aureus*, *Streptococcus agalactiae*, groups A, F and G streptococci, gram-positive organisms, and some gram-negative organisms if pretreated with a chelator (Boddie and Nickerson, 1992; Kabara, 1978, 1984; Isaacs et al., 1990, 1992, 1994; Isaacs and Schneidman, 1991; Isaacs and Thormar, 1986, 1990, 1991; Thormar et al., 1987; Wang and Johnson, 1992).

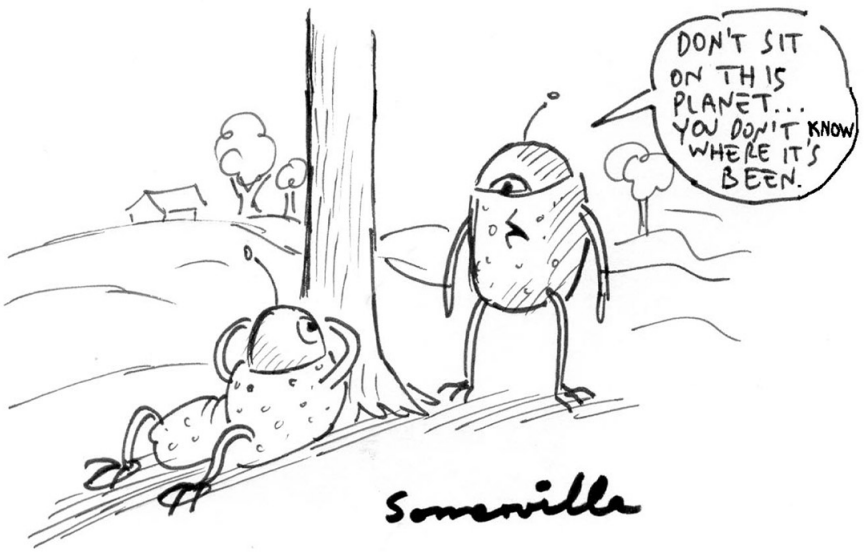
Decreased growth of *Staphylococcus aureus* and decreased production of toxic shock syndrome toxin-1 was shown with 150 mg monolaurin per litre (Holland et al., 1994). Monolaurin was shown to be 5,000 times more inhibitory against *Listeria monocytogenes* than is ethanol (Oh and Marshall, 1993). *Helicobacter pylori* was rapidly inactivated by medium-chain monoglycerides and lauric acid, and there appeared to be very little development of resistance of the organism to the bactericidal effects of these natural antimicrobials (Petschow et al., 1996).

A number of fungi, yeast and protozoa have been found to be inactivated or killed by lauric acid or monolaurin. The fungi include several species of ringworm (Isaacs et al., 1991). The yeast reported is *Candida albicans* (Isaacs et al., 1991). The protozoan parasite *Giardia lamblia* is killed by free fatty acids and monoglycerides from hydrolysed human milk (Hernell et al., 1986; Reiner et al., 1986; Crouch et al., 1991; Isaacs et al., 1991). Numerous other protozoa were studied with similar findings, but these have not yet been published (Jon J. Kabara, private communication, 1997).

Research continues in measuring the effects of the monoglyceride derivative of capric acid, monocaprin, as well as the effects of lauric acid. *Chlamydia trachomatis* is inactivated by lauric acid, capric acid and monocaprin (Bergsson et al., 1998). Hydrogels containing monocaprin are potent *in vitro* inactivators of sexually transmitted viruses such as HSV-2 and HIV-1 and bacteria such as *Neisseria gonorrhoeae* (Thormar, 1999).

III. ORIGINS OF THE ANTI–SATURATED FAT, ANTI–TROPICAL OILS AGENDA

The coconut industry has suffered more than three decades of abusive rhetoric from the consumer activist group Centers for



Science in the Public Interest (CSPI), from the American Soybean Association (ASA) and other members of the edible oil industry, and from those in the medical and scientific community who learned their misinformation from groups like CSPI and ASA. I would like to review briefly the origins of the anti–saturated fat, anti–tropical oil campaigns and hopefully give you some useful insight into the issues.

When and how did the anti–saturated fat story begin? It really began in part in the late 1950s, when a researcher in Minnesota announced that the heart disease epidemic was being caused by hydrogenated vegetable fats. The edible oil industry's response at that time was to claim it was only the saturated fat in the hydrogenated oils that was causing the problem. The industry then announced that it would be changing to partially hydrogenated fats and that this would solve the problem.

In actual fact, there was no change because the oils were already being partially hydrogenated and the levels of saturated fatty acids remained similar, as did the levels of the *trans* fatty acids. The only thing that really changed was the term for "hydrogenation" or "hardening" listed on the food label.

During this same period, a researcher in Philadelphia reported that consuming polyunsaturated fatty acids lowered serum cholesterol. This researcher neglected, however, to include the information that the lowering was due to the cholesterol going into the tissues such as the liver and the arteries. As a result of this research report and the acceptance of this new agenda by the domestic edible oils industry, there was a gradual increase in the emphasis on replacing "saturated fats" in the diet and on consuming larger amounts of the "polyunsaturated fats".

As many of you probably know, this strong emphasis on consuming polyunsaturates has backfired in many ways.

As many of you probably know, this strong emphasis on consuming polyunsaturates has backfired in many ways. The current adjustments, being recommended in the US by groups such as the National Academy of Sciences, replace the saturates with mono-unsaturates instead of with polyunsaturates and replace polyunsaturates with mono-unsaturates.

Early promoters of the anti–saturated fat ideas included companies such as Corn Products Company (CPC International), through a book written by Jeremiah Stamler in 1963, with the professional edition published in 1966 by CPC. This book took some of the earliest pejorative stabs at the tropical oils. In 1963, the only tropical fat or oil singled out as high in saturated fats was coconut oil. Palm oil had not entered the US food supply to any extent, had not become a commercial threat to the domestic oils and was not recognised in any of the early texts.

The editorial staff of *Consumer Reports* noted that "...in 1962...one writer observed, the average American now fears fat [saturated fat, that is] 'as he once feared witches'".

In 1965, a representative of Procter & Gamble Pharmaceuticals told the American Heart Association to change its diet/heart statement to remove any reference to the *trans* fatty acids. This altered official document encouraged the consumption of partially hydrogenated fats. In the 1970s, this same Procter & Gamble employee served as nutrition chairman in two controlling positions for the National Heart, Lung, and Blood Institute's Lipid Research Clinic (LRC) trials and as director of one of the LRC centres. These LRC trials were the basis for the 1984 NIH Cholesterol Consensus Conference, which in turn spawned the National Cholesterol Education Program (NCEP). This program encourages consumption of margarine and partially hydrogenated fats, while admitting that *trans* should not be consumed in excess. The official NCEP document states that "coconut oil, palm oil, and palm kernel oil...should be avoided".

In 1966, the US Department of Agriculture documents on fats and oils talked about how unstable the unsaturated fats and oils were. There was no criticism of the saturated fats. That criticism of saturated fats was to come later to this agency when it came under the influence of the domestic edible fats and oils industry and when it developed the US Dietary Guidelines. These Dietary Guidelines became very anti–saturated fat and remain so to this day. Nevertheless, as we will learn later in my talk, there started some reversal of the anti–saturated fat stance in the works of this agency in 1998.

In the early 1970s, although a number of researchers were voicing concerns about the *trans* fats, the edible oil industry and the US Food and Drug Administration (FDA) were engaging in a revolving-door exchange that would promote the increasing consumption of partially hydrogenated vegetable oils, condemn the saturated fats and hide the *trans* issue. As an example of this "oily" exchange, in 1971 the FDA's general counsel became president of the edible oil trade association, the Institute of Shortening and Edible Oils (ISEO), and he in turn was replaced at the FDA by a food lawyer who had represented the edible oil industry.

From that point on, the truth about any real effects of the dietary fats had to play catch-up. The American edible oil industry sponsored "information" to educate the public, and the natural dairy and animal fats industries were inept at countering any of that misinformation. Not being domestically grown in the US, coconut oil, palm oil and palm kernel oil were not around to defend themselves at that time. The government agencies responsible for disseminating information ignored those protesting "lone voices", and by the mid-1980s American food manufacturers and consumers had made major changes in their fats and oils usage—away from the safe, saturated fats and headlong into the problematic *trans* fats.

Enig and Fallon (1998–99) have reviewed the above history in "The Oiling of America", published in NEXUS Magazine [see 6/01–2]. This article can be viewed and downloaded from the NEXUS website at www.nexusmagazine.com/OilingAmerica.1.html and www.nexusmagazine.com/OilingAmerica.2.html.

IV. THE DAMAGING ROLE OF THE US CONSUMER ACTIVIST GROUP CSPI

Some of the food oil industry members—especially those connected with the American Soybean Association and some of the consumer activists (particularly the Centers for Science in the Public Interest and also the American Heart Savers Association) further eroded the status of natural fats when they sponsored the major anti–saturated fat, anti–tropical oils campaign in the late 1980s.

Actually, an active anti–saturated fat bias started as far back as 1972 at the CSPI. But beginning in 1984, this very vocal consumer activist group started its anti–saturated fat campaign in earnest. In particular at this time, the campaign was against the "saturated" frying fats, especially those being used by fast-food restaurants. Most of these so-called saturated frying fats were tallow-based, but also included was palm oil in at least one of the hotel/restaurant chains.

Then, in a critical "News Release" in August 1986—"Deceptive Vegetable Oil Labeling: Saturated Fat Without The Facts"—CSPI referred to "palm, coconut and palm kernel oil" as "rich in artery-clogging saturated fat". CSPI further announced that it had petitioned the Food and Drug Administration to stop allowing labelling of foods as having "100% vegetable shortening" if they contained any of the "tropical oils". CSPI also asked for the mandatory addition of the qualifier, "a saturated fat", when coconut, palm or palm kernel oil was named on the food label.

In 1988, CSPI published a booklet called "Saturated Fat Attack". This booklet contains lists of processed foods "surveyed" in Washington, DC, supermarkets. The lists were used for developing information about the saturated fat in the products. Section III is entitled "Those Troublesome Tropical Oils" and it contains statements encouraging pejorative labelling. There were lots of substantive mistakes in the booklet, including errors in the description of the biochemistry of fats and oils and completely erroneous statements about the fat and oil composition of many of the products.

At the same time that CSPI was conducting its campaign in 1986, the American Soybean Association began its anti–tropical oils campaign by sending inflammatory letters, etc., to soybean farmers. The ASA took out advertisements to promote a "[tropical] Fat Fighter Kit". The ASA hired a Washington, DC, "nutritionist" to survey supermarkets to detect the presence of tropical oils in foods.

Then, early in 1987, the ASA petitioned the FDA to require labelling of "tropical fats". In mid-1987 the *Soybean Digest* was continuing an active and increasing anti–tropical oils campaign.

At about the same time, the *New York Times* (June 3, 1987) published an editorial, "The Truth About Vegetable Oil", in which it called palm, palm kernel and coconut oils "the cheaper, artery-clogging oils from Malaysia and Indonesia" and claimed that US federal dietary guidelines opposed tropical oils, although it is not clear that this was so. The "artery-clogging" terminology was right out of CSPI.

Two years later, in 1989, the ASA held a press conference with the help of the CSPI in Washington, DC, in an attempt to counter a press conference held on March 6 by the palm oil group. The ASA "Media Alert" stated that the National Heart, Lung, and Blood Institute and National Research Council "recommend consumers avoid palm, palm kernel and coconut oils".

Only months before these press conferences, millionaire Phil Sokolof, the head of the National Heart Savers Association (NHSA), purchased the first of a series of anti–saturated fats and anti–tropical fats advertisements in major newspapers. No one has found an overt connection between Sokolof (and his NHSA) and the ASA, but the CSPI bragged about being his adviser.

V. USE OF COCONUT OIL IN THE PREVENTION AND TREATMENT OF HEART DISEASE

The research over four decades concerning coconut oil in the diet and heart disease is quite clear: coconut oil has been shown to be beneficial in combatting/reducing the risk factors in heart disease. This research leads us to ask the question, "Should coconut oil be used both to prevent and treat coronary heart disease?" This is based on several reviews of the scientific literature concerning the feeding of coconut oil to humans.

The research over four decades concerning coconut oil in the diet and heart disease is quite clear: coconut oil has been shown to be beneficial in combatting/reducing the risk factors in heart disease.

Blackburn et al. (1988) reviewed the published literature of "coconut oil's effect on serum cholesterol and atherogenesis" and concluded that when "fed physiologically with other fats or adequately supplemented with linoleic acid, coconut oil is a neutral fat in terms of atherogenicity".

After reviewing this same literature, Kurup and Rajmohan (1995) conducted a study on 64 volunteers and found "no statistically significant alteration in the serum total cholesterol, HDL cholesterol, LDL cholesterol, HDL cholesterol/total cholesterol ratio and LDL cholesterol/HDL cholesterol ratio of triglycerides from the baseline values". A beneficial effect of adding the coconut kernel to the diet was noted by these researchers.

Kaunitz and Dayrit (1992) reviewed some of the epidemiological and experimental data regarding coconut-eating groups and noted that the "available population studies show that dietary coconut oil does not lead to high serum cholesterol nor to high coronary heart disease mortality or morbidity".

They noted that, in 1989, Mendis et al. reported undesirable lipid changes when young adult Sri Lankan males were changed from their normal diets by the substitution of corn oil for their customary coconut oil. Although the total serum cholesterol decreased 18.7% from 179.6 to 146.0 mg/dL and the LDL cholesterol decreased 23.8% from 131.6 to 100.3 mg/dL, the HDL cholesterol decreased 41.4% from 43.4 to 25.4 mg/dL (putting the HDL values very much below the acceptable lower limit of 35 mg/dL) and the LDL/HDL ratio increased 30% from 3.0 to 3.9. These latter two changes are considered quite undesirable.

Mendis and Kumarasunderam (1990) also compared the effect of coconut oil and soy oil in normolipidemic young males, and again the coconut oil resulted in an increase in the HDL cholesterol, whereas the soy oil reduced this desirable lipoprotein.

As noted above, Kurup and Rajmohan (1995), who studied the addition of coconut oil alone to previously mixed fat diets, had reported no significant difference from baseline.

Previously, Prior et al. (1981) had shown that islanders with high intakes of coconut oil showed "no evidence of the high saturated fat intake having a harmful effect in these populations". When these groups migrated to New Zealand, however, and lowered their intake of coconut oil, their total cholesterol and LDL cholesterol increased and their HDL cholesterol decreased. Statements that any saturated fat is a dietary problem is not supported by evidence (Enig, 1993).

Studies that allegedly showed a "hypercholesterolemic" effect of coconut oil feeding usually only showed that coconut oil was not as effective at lowering the serum cholesterol as was the more unsaturated fat to which coconut oil was being compared. This appears to be in part because coconut oil does not "drive" cholesterol into the tissues as do the more polyunsaturated fats. The chemical analysis of the atheroma showed that the fatty acids from the cholesterol esters are 74% unsaturated (41% of the total fatty acids is polyunsaturated) and only 24% are saturated. None of the saturated fatty acids was reported to be lauric acid or myristic acid (Felton et al., 1994).

Thus, ironically enough, one could consider the recommendations to avoid coconut and other lauric oils as *contributing* to the increased incidence of coronary heart disease!

There is another aspect to the coronary heart disease picture. This is related to the initiation of the atheromas that are reported to be blocking arteries. Recent research shows that there is a causative role for the herpes virus and cytomegalovirus in the initial formation of atherosclerotic plaques and the reclogging of arteries after angioplasty (*New York Times*, January 29, 1991). What is so interesting is that the herpes virus and cytomegalovirus are both inhibited by the antimicrobial lipid monolaurin, but monolaurin is not formed in the body unless there is a source of lauric acid in the diet.

Thus, ironically enough, one could consider the recommendations to avoid coconut and other lauric oils as contributing to the increased incidence of coronary heart disease.

Chlamydia pneumoniae, a gram-negative bacterium, is another of the micro-organisms suspected of playing a role in atherosclerosis by provoking an inflammatory process that would result in the oxidation of lipoproteins with induction of cytokines and production of proteolystic enzymes—a typical phenomenon in atherosclerosis (Saikku, 1997). Some of the pathogenic gram-negative bacteria with an appropriate chelator have been reported to be inactivated or killed by lauric acid and monolaurin as well as capric acid and monocaprin (Bergsson et al., 1997; Thormar et al., 1999).

However, the micro-organisms which are most frequently identified as probable causative infecting agents are in the herpes virus family and include cytomegalovirus, type 2 herpes simplex (HSV-2) and Coxsackie B4 virus.

The evidence for a causative role for cytomegalovirus is the strongest (Ellis, 1997; Visseren et al., 1997; Zhou et al., 1996; Melnick et al., 1996; Epstein et al., 1996; Chen and Yang, 1995), but a role for HSV-2 is also shown (Raza-Ahmad et al., 1995).

All members of the herpes virus family are reported to be killed by the fatty acids and monoglycerides from saturated fatty acids ranging from C-6 to C-14 (Isaacs et al., 1991), which include approximately 80% of the fatty acids in coconut oil.

In spite of what has been said over the past four or more decades about the culpability of the saturated fatty acids in heart disease, they are ultimately going to be held blameless. More and more research is showing the problem to be related to oxidised products. The naturally saturated fats such as coconut oil are one protection we have against oxidised products.

Continued in the next issue of NEXUS Magazine

Editor's Note:
As we have insufficient space to publish the numerous references accompanying this article, we have instead posted them on the NEXUS Magazine website, www.nexusmagazine.com.

About the Author:
Dr Mary G. Enig holds an MS and PhD in Nutritional Sciences from the University of Maryland in the USA. She is a consulting nutritionist and biochemist of international renown and an expert in fats/oils analysis and metabolism, food chemistry and composition and nutrition and dietetics.

Dr Enig is Director of the Nutritional Sciences Division of Enig Associates, Inc., President of the Maryland Nutritionists Association and a Fellow of the American College of Nutrition. She is also Vice President of the Weston A. Price Foundation and Science Editor of the Foundation's publication. Dr Enig has many years of experience as a lecturer and has taught graduate-level courses for the Nutritional Sciences Program at the University of Maryland, where she was a Faculty Research Associate in the Lipids Research Group, Department of Chemistry and Biochemistry, University of Maryland. She also maintains a limited clinical practice for patients needing nutritional assessment and consultation.

Dr Enig has extensive experience consulting and lecturing on nutrition to individuals, medical and allied health groups, the food processing industry and state and federal governments in the US. She also lectures and acts as a consultant to the international health and food processing communities. Since 1995 she has been invited to make presentations at scientific meetings in Europe, India, Japan, Vietnam, Indonesia, the Philippines and Micronesia.

Dr Enig is the author of numerous journal publications, mainly on fats and oils research and nutrient/drug interactions. She also wrote the book *Know Your Fats* (Bethesda Press, Silver Spring, MD, May 2000). She is a popular media spokesperson and was an early critic speaking out about the use of *trans* fatty acids and advocating their inclusion in nutritional labelling.

One of Dr Enig's recent research topics dealt with the development of a nutritional protocol for proposed clinical trials of a non-drug treatment for HIV/AIDS patients. Her articles, "The Oiling of America" and "Tragedy and Hype: The Third International Soy Symposium", written with nutritionist/researcher Sally Fallon, were published in NEXUS 6/01–2 and 7/03 respectively.